Supplemental Figure 1. CD19 and B220 staining in e16.5 fetal liver. Unstained, isotype control, and KO are shown as indicated. Marking on histogram scores population designated as "positive" to establish gates used in Fig. 4.

Supplemental Figure 2. SXR modulates NF-κB targets in $CD19^+$ fetal liver cells during embryonic development. qPCR analysis of indicated transcripts from magnetically purified $CD19^+$ B-1 cell precursors from fetal livers resected from WT (n=12) or KO (n=7) e16.5 embryos is shown, and the means are indicated. Data are shown as fold of WT, and statistics are relative to WT.

Supplemental Figure 3. SXR modulates B-1 cell markers in $CD19^+$ fetal liver cells during embryonic development. qPCR analysis of indicated transcripts from magnetically purified $CD19^+$ B-1 cell precursors from fetal livers resected from WT (n=12) or KO (n=7) e16.5 embryos is shown, and the means are indicated. Data are shown as fold of WT, and statistics are relative to WT.

Supplemental Figure 4. In utero activation of SXR by 5.1 μM PCN as determined by target gene induction. A-B: Expression of CYP3A11 and CYP3A16 in e16.5 fetal liver. Dams were treated with CMC or PCN from e0.5 until embryos were harvested and fetal livers resected at e16.5. CMC, n=4, PCN, n=3. Expression is relative to GAPDH and is expressed as fold of CMC. C: Expression of CYP3A11 in 8 week liver. Dams were treated with CMC or PCN from e0.5 until birth, at which point treatment was removed. CMC, n=3, PCN, n=4. Expression is relative to GAPDH and is expressed as fold of CMC.

Supplemental Figure 5: Loss of SXR increases B-1 cell progenitors in the developing fetal liver whereas SXR activation reduces B-1 cell progenitors. A-C: Dams were treated with mouse SXR agonist PCN (at μM indicated) or vehicle control from conception until e16.5. Fetal livers were resected, dissociated, and stained. Cells that stained $CD19^{-}/B220^{+}$, $CD19^{+}/B220^{+}$ or $CD19^{+}/B220^{low}$ are indicated. Percentage of total cells residing in each gate was quantitated for each condition (Fig. 4).

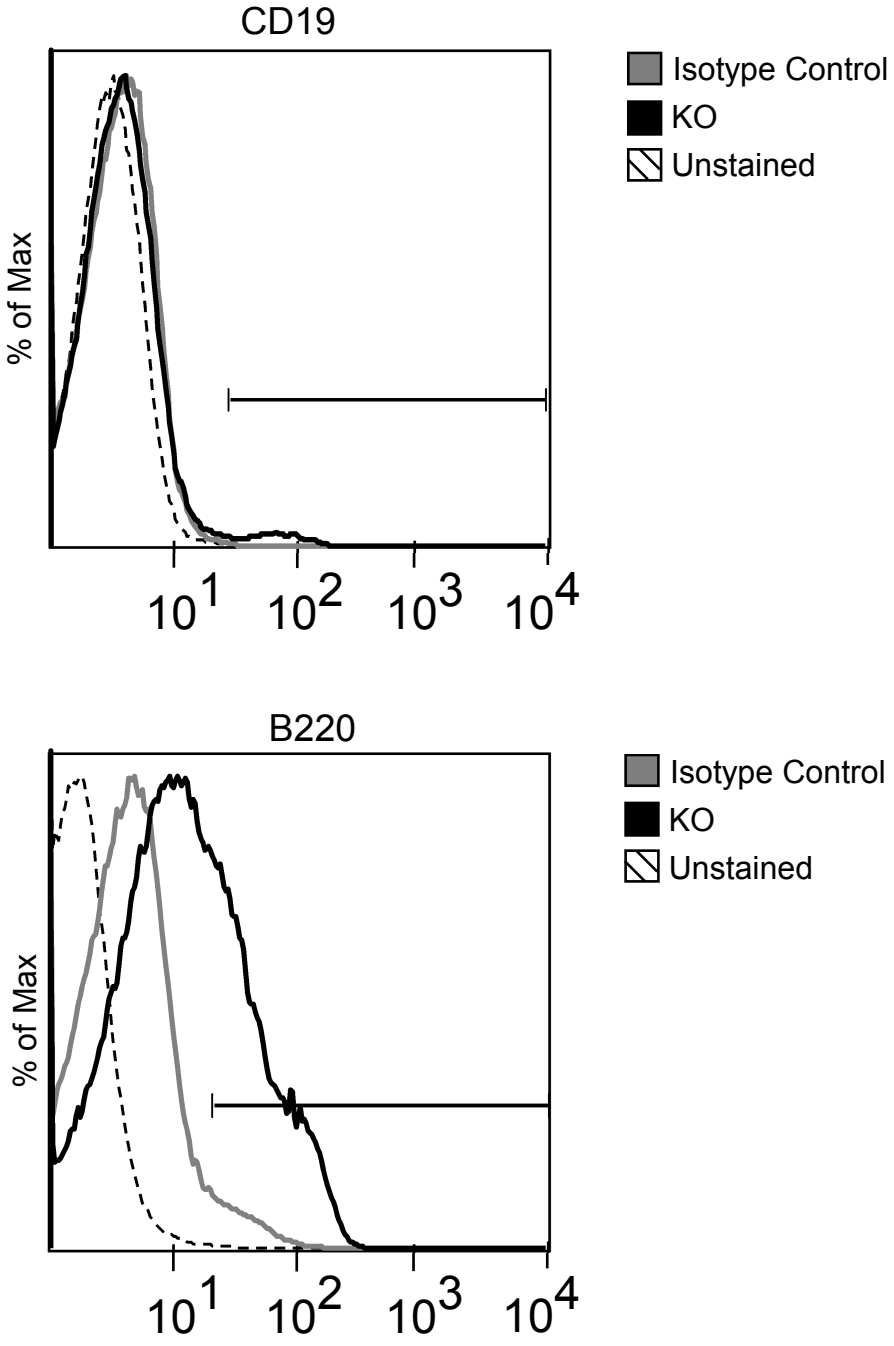
Supplemental Figure 6. In utero exposure to PCN does not affect spleen weight (in mg). Data are shown from animals examined at 8 weeks of age, and mean is indicated.

Supplemental Figure 7: In Utero exposure to murine SXR agonist PCN reduces the size of the B-1 cell compartment. Cells collected from unchallenged peritoneal lavage from animals treated in utero with CMC or PCN were analyzed for the expression of IgM and CD5. Gate indicates B-1 cells ($IgM^{+}/CD5^{+}$). The percentage of cells that stained positive as determined by flow cytometry was quantitated and graphed in Fig. 5.

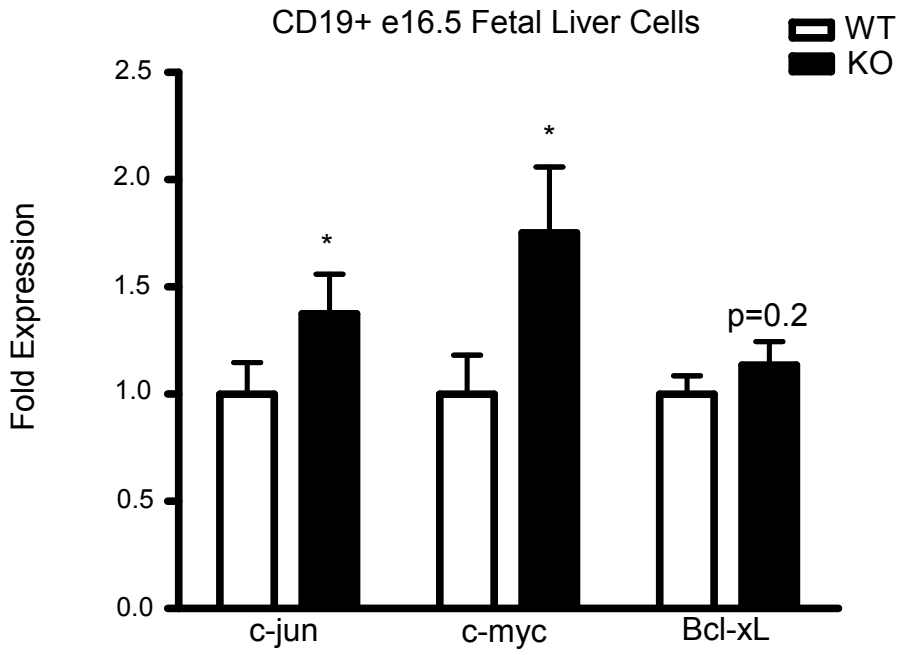
Supplemental Table 1: Primer sequences used for qPCR.

Supplemental Figure 1

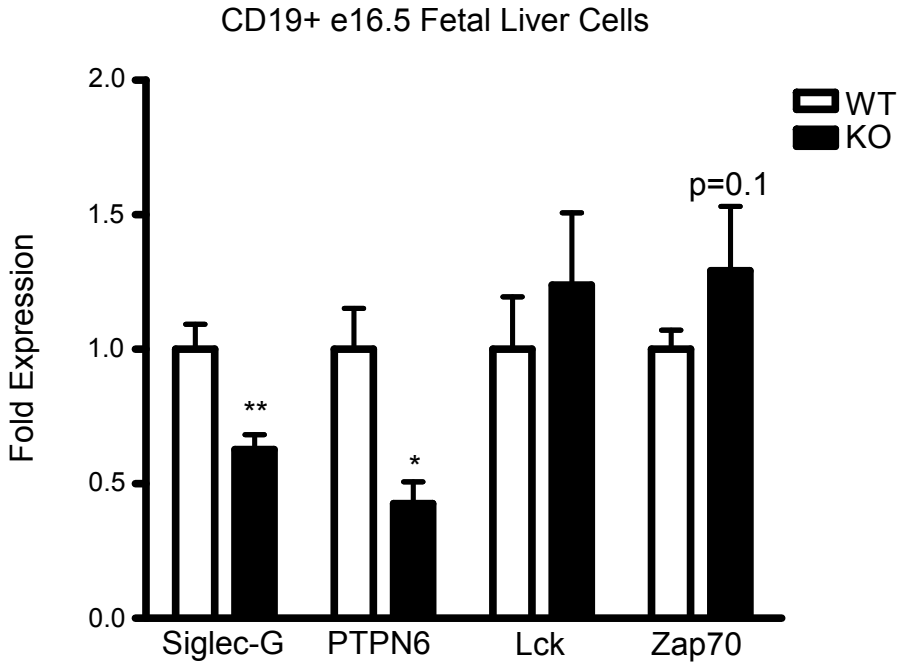
Flow data for Fig 4A



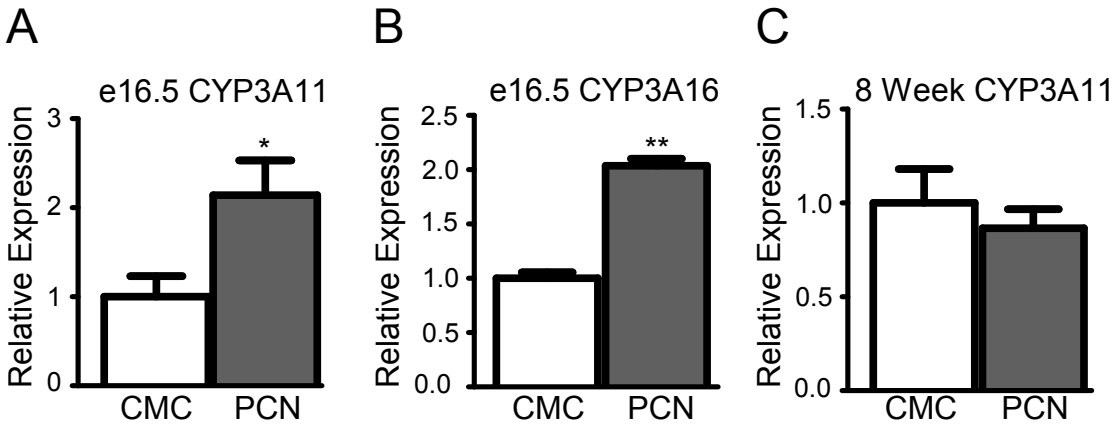
Supplemental Figure 2



Supplemental Figure 3

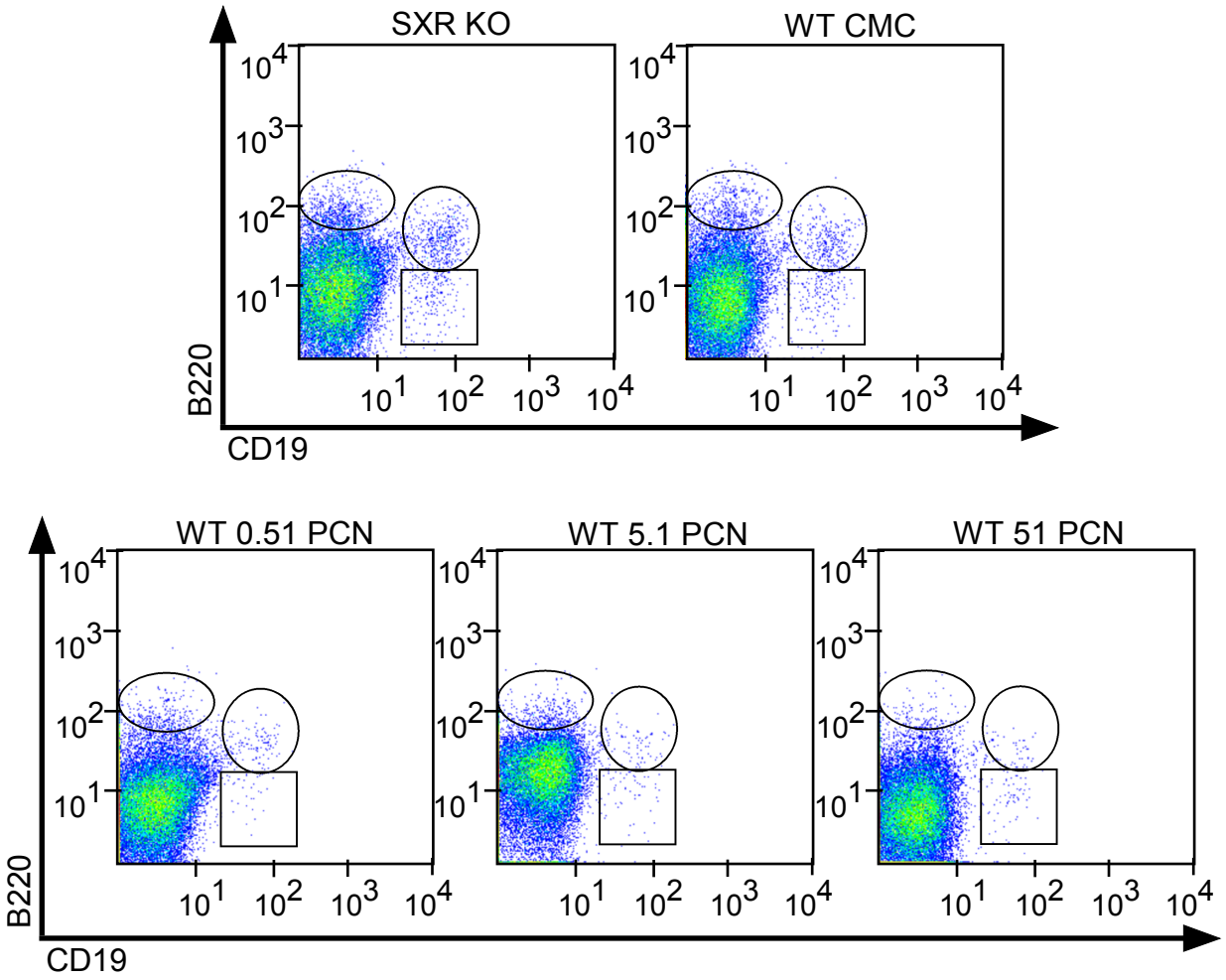


Supplemental Figure 4

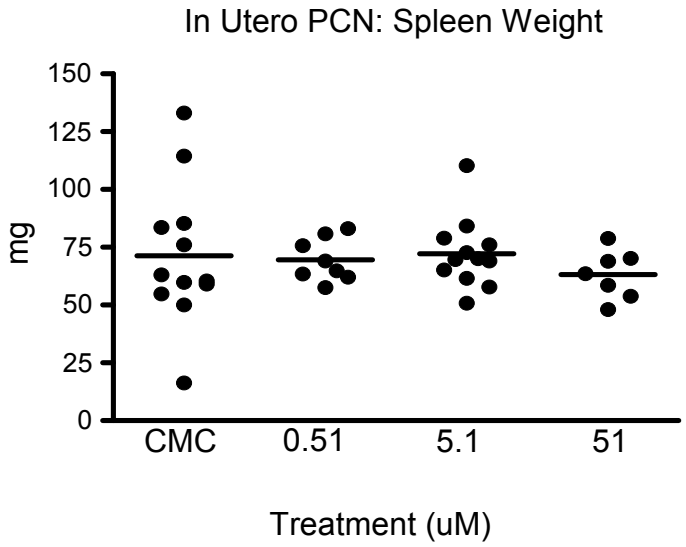


Supplemental Figure 5

Representative Flow Plots of
PCN Treated Progenitor Cells

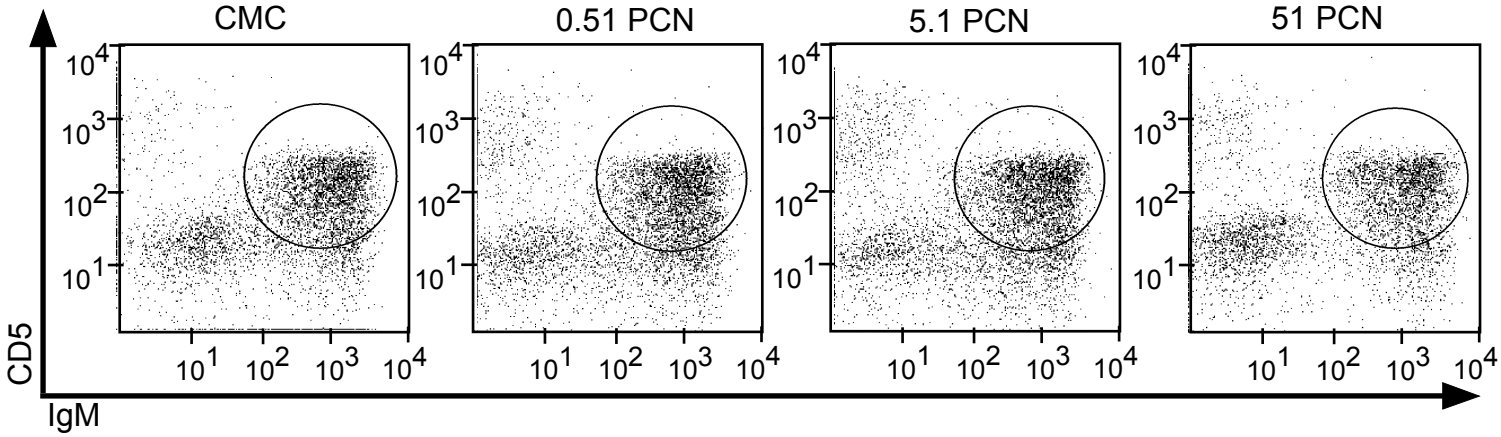


Supplemental Figure 6



Supplemental Figure 7

Flow Graphs of PCN Peritoneal Lavages



Supplemental Table 1

Gene	Forward Primer	Reverse Primer
Bcl-xL	GGAGAGCGTTCAGTGATC	GGTGGTCATTCAGATAGG
c-jun	GCCAACCTCAGCAACTTC	GCTTCCTCTCTGCCTTGA
c-myb	ATTGTGGACCAGACCAGACC	GCTGGTGAGGCACTTTCTTC
c-myc	TGAGCCCCTAGTGCTGCAT	AGCCCGACTCCGACCTCTT
CYP3A11	CAGCTTGGTCCTCCTCTACC	TCAAACAACCCCCATGTTTT
CYP3A16	CCACCAGCAGCATACTTTCCT	CCATCGCCATCACGGTATCATA
GAPDH	AGGTCGGTGTGAACGGATTTG	TGTAGACCATGTAGTTGAGGTCA
Lck	CCACCTCACAATCCCAGCAG	CCTCGGGGAGGGTTCATTC
PTPN6	GCAGGAGAACACTCGTGTCA	CCCATTGTCTAGTGGGGAGA
Siglec-G	GCCTCAAGGTCAGATGGAGA	AGGCTCCAGGACCTCAGGAA
SXR	GACGCTCAGATGCAAACCTT	TGGTCCTCAATAGGCAGGTC
Zap-70	GGGGTCTTCGACTGCCTGCG	GCCTGGCTGATGATGGCCTGC